

RESEARCH PAPER

Relationship between sarcopenia and orthostatic hypotension

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Abstract

Background: The relationship between sarcopenia and orthostatic hypotension (OH) is unclear.

Objectives: The aim of the present study was to investigate associations between sarcopenia/sarcopenia severity and OH.

Design: A total of 511 patients attending a geriatric outpatient clinic were included. OH was defined as a decrease in systolic and/or diastolic blood pressure of ≥ 20 mmHg and/or ≥ 10 mmHg, respectively, when one transitions from the supine to an upright position. OH was measured by the Head-up Tilt Table test at 1, 3 and 5 min (OH₁, OH₃ and OH₅, respectively). Sarcopenia and its severity were defined according to the revised European consensus on definition and diagnosis.

Results: The mean age of the sample was 75.40 ± 7.35 years, and 69.9% were female. The prevalence of probable sarcopenia, sarcopenia and severe sarcopenia was 42.2%, 6.06% and 11.1%, respectively. After adjustment for all covariates, systolic OH₁, OH₁ and systolic OH₅ were statistically significantly different between severe sarcopenia and the robust group (odds ratio [OR]: 3.26, confidence interval [CI] 0.98–10.84; $P = 0.05$ for systolic OH₁; OR 4.31, CI 1.31–14.15; $P = 0.016$ for OH₁; OR 4.09, CI 1.01–16.55; $P = 0.048$ for systolic OH₅). Only systolic OH₁ was statistically different between the sarcopenia and severe sarcopenia groups (OR 2.64, CI 1.87–8.73; $P = 0.012$). OH₁ and OH₅ were statistically significant different between severe sarcopenia and probable sarcopenia groups ($P < 0.05$); there was no relationship between the robust group and probable sarcopenia ($P > 0.05$).

Conclusions: There is a close relationship between sarcopenia and severe sarcopenia and OH in older adults. Therefore, when a healthcare practitioner is evaluating an older patient with sarcopenia, OH should also be evaluated, and vice versa.

Keywords: sarcopenia, orthostatic hypotension, geriatric assessment, older people

Key points

- There is an association between sarcopenia and orthostatic hypotension.
- The greater the severity of sarcopenia, the stronger the association is with orthostatic hypotension.
- Decreased muscle mass, rather than muscle strength, is associated with orthostatic hypotension.
- Orthostatic hypotension and sarcopenia are two major geriatric syndromes.

Introduction

With advancing age, there is a significant decrease in compensatory organ functions that provide homeostatic balance in the body [1]. Orthostatic (postural) hypotension (OH)

is one health consequence resulting from a deterioration in homeostasis [1]. While the prevalence of OH is less than 5% before the age of 65 years, it is stated that this rate exceeds 30% for those aged 70 years and over [1]. OH causes cardiovascular events, recurrent falls, depression, stroke,

cognitive impairment and mortality in geriatric patients [2–5]. Recently, it has been argued that OH may be a new geriatric syndrome rather than a cardiovascular condition [4]. Therefore, early detection of risk factors for developing OH is important.

OH is defined as a decrease in systolic blood pressure of ≥ 20 mmHg and/or a decrease in diastolic blood pressure of 10 mmHg in the first 3 min of transition from the supine position to the upright position [6]. While the patient is standing up, a normal rise in systolic blood pressure of 5–10 mmHg and a rise in diastolic blood pressure of 5–10 mmHg are considered physiological, termed orthostatic stability in healthy individuals, and are a consequence of neurohumoral and venous pump activation (muscle contractions) over approximately 60 s [1,7]. However, decreased arterial baroreceptor sensitivity and renin–angiotensin–aldosterone levels through ageing, cardiac hypertrophy and the failure of the autonomic nervous system increase the risk of developing OH [7]. One mechanism contributing to the development of OH is a decrease in venous circulation of blood pooled in the lower half of the body owing to gravity [8]. The mechanism is impaired as a result of the inadequate response of the skeletal muscle pumps to changes in the circulatory system, and the decrease in skeletal muscle contraction of the legs in older adults, leading to the hypothesis that sarcopenia, an important geriatric syndrome, is likely to cause OH.

This hypothesis has also been indirectly supported by some studies. For example, research has shown that sarcopenia, sarcopenic obesity and low muscle mass may increase the risk of developing OH by increasing insulin resistance and inflammation, and causing arterial stiffness [9]. For example, Zhang *et al.* reported that high brachial–ankle pulse wave velocity, indicative of arterial stiffness, was associated with sarcopenia [9]. In another study, reduced muscle mass was an independent risk factor for increased arterial stiffness [10]. Indeed, arterial stiffness leads to a decrease in the sensitivity of baroreceptors that control arterial blood pressure, causing OH [11]. Moreover, one of the most important causes of OH is autonomic dysfunction, and recent studies have shown that there is impairment in muscle sympathetic nerve activity due to reduced muscle mass in sarcopenia, leading to sympatho-vagal imbalance and OH [12,13].

In recent years it has been reported that OH may be caused by frailty and malnutrition, which are closely associated with sarcopenia [14,15]. However, to the best of our knowledge, no study has been conducted to date to examine the relationship between sarcopenia and OH. For this reason, the aim of the present study was to investigate associations between sarcopenia and its severity with OH.

Methods

Inclusion criteria

A total of 849 older patients attended (referred by a clinician or self-referred), for any health issue, one outpatient clinic

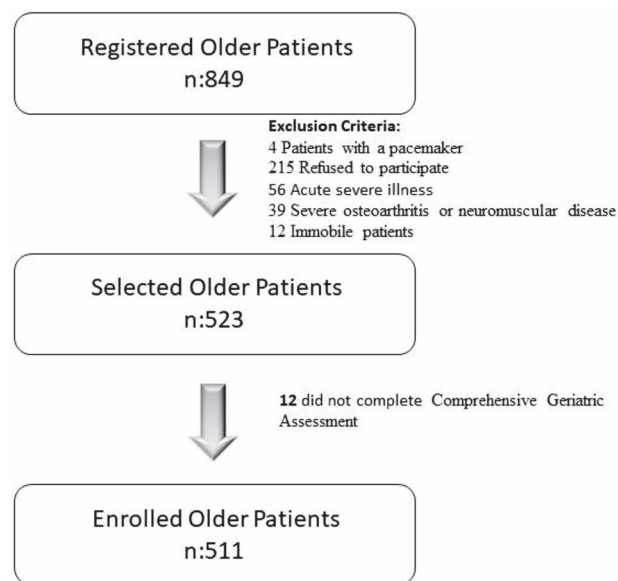


Figure 1. Prisma flow chart.

between September 2016 and October 2018. This clinic is located in the department of geriatric medicine at the university teaching hospital, Dokuz Eylul University in Izmir, Turkey.

A total of 511 patients without any exclusion criteria were included in this cross-sectional study (Figure 1). The investigation conformed to the Declaration of Helsinki and was approved by the local ethics committee. Informed consent was provided by each participant or a legal surrogate.

Exclusion criteria

The following groups of patients were excluded. (i) patients with severe osteoarthritis or neuromuscular disease, which causes an obstacle to walking, and immobile patients; (ii) patients with any contraindications for the Head up Tilt Table test (HUT) [14]; (iii) patients with alcohol and substance abuse; (iv) patients with a pacemaker (because of contraindication to electrical bioimpedance); and (v) patients under 65 years of age and those who refused to participate.

Patient characteristics

Patients' age, gender, level of education, concurrent systemic and chronic diseases, and number of medications used were determined by patients' self or caregiver report. During admission, they were asked whether they had fallen in the previous year. Dementia and depression were diagnosed according to Diagnostic and Statistical Manual of Mental Disorders Fifth Edition (DSM-5) criteria.

Comprehensive geriatric assessment

Patients had a specialist evaluation by a geriatrician, during which the following assessment scales were routinely completed: Mini Mental State Examination (MMSE), Yesevage

Geriatric Depression Scale (GDS) for neurocognitive evaluation, Basic and Instrumental Activities of Daily Living (BADL and IADL) for functionality evaluation, Timed Up and Go test and Tinetti Performance-Oriented Assessment of Mobility (POMA) for mobility evaluation, and Mini Nutritional Assessment (MNA) for nutritional evaluation [16].

Laboratory findings

Laboratory tests, including renal and liver function, fasting blood glucose, haemogram, thyroid-stimulating hormone (TSH), vitamin D, vitamin B12 and folic acid levels, were performed to evaluate the biochemical, metabolic and nutritional status of the patients, all of which were obtained with the auto-analyser diagnostic modular system (Roche E170 and P-800). Serum 25-hydroxy D vitamin [25(OH)D] was measured by radioimmunoassay.

Diagnosis of sarcopenia

For the evaluation of walking speed, muscle strength and muscle mass, 4 m walking test, handgrip test and bioimpedance, respectively, were performed for each patient. Handgrip test was measured by a JAMAR branded hand dynamometer, and bioimpedance was established using TANITA scales (MC-780 U Multi Frequency Segmental Body Composition). Slow walking speed was categorized as <0.8 m/s, and low handgrip strength in women as <16 kg and in males as <27 kg [17]. A muscle mass index score of <8.87 kg/m² for males and of <6.42 kg/m² for females was regarded as low muscle mass [17]. Probable sarcopenia, sarcopenia and severe sarcopenia were identified according to revised European consensus on definition and diagnosis (EWGSOP) criteria [17,18]. The robust group included those patients without probable sarcopenia, sarcopenia and severe sarcopenia.

Diagnosis of OH

OH was evaluated using HUT [14]. HUT was conducted using the Tilt Table (Gemesan1 Tilt TableG-71, Turkey). Monitoring over the course of HUT was performed using the Biolight1 BIOM69 (Australia). The patients were monitored for blood pressure over the course of HUT. The data were obtained from measurements recorded in the first, third and fifth minutes. Continuous beat-to-beat blood pressure measurements were not obtained. OH was defined as a decrease in systolic and/or diastolic blood pressure of ≥ 20 mmHg and/or ≥ 10 mmHg, respectively, when one transitions from a supine to an upright position. For example, the diagnosis of OH in the first minute was made by the presence of systolic OH in the first minute and/or diastolic OH in the first minute.

Statistical analyses

Data were analysed using SPSS for Windows 15. Descriptive statistics are shown as mean \pm standard deviation for

variables with normal distribution, median (minimum to maximum) for non-normal distributions and the number of cases and percentage (%) for nominal variables. When the group number was two, the significance of differences between the groups in terms of averages was investigated by *t*-test and in terms of median values was investigated by Mann–Whitney test. When the number of groups was more than two or the variables were not normally distributed, the significance of medians was determined by the Kruskal–Wallis test. Nominal variables were assessed by Pearson χ^2 or Fisher exact test. Binary logistic regression analysis was performed within two groups adjusted for confounding variables. Odds ratios (ORs) were calculated. $P < 0.05$ was considered statistically significant. In a review carried out by Frith *et al.* in 2017, it was identified that the frequency of OH in community-dwelling people aged ≥ 65 years was 30%. Therefore, at least 269 patients were required to be recruited into the present study, to achieve a level of 5% acceptable error and 95% confidence [19].

Results

The prevalence of probable sarcopenia, sarcopenia and severe sarcopenia was 42.2%, 6.06% and 11.1%, respectively. Of 511 older patients, 69.9% were female and the mean age was 75.40 ± 7.35 years. Age, gender, education year, body mass index (BMI), falls history, presence of dementia, level of haemoglobin, albumin, MMSE, YGDS, Tinetti-gait and balance scores, Timed up and go test score, ADLs and MNA score were statistically different within groups according to revised EWGSOP criteria ($P < 0.05$) (Table 1). Additionally, the number of drugs used, estimated glomerular filtration rate (eGFR) and level of 25(OH)D were statistically significantly different within groups ($P < 0.05$, Table 1). The rates of systolic OH₁, OH₁ and OH₅ were different within groups ($P < 0.05$, Table 1).

In binary logistic regression analysis, adjusted for age, BMI, gender, education year, falls history, presence of dementia, MMSE, YGDS, Tinetti-gait, balance and total test, Timed up and go test score, ADLs (Barthel and Lawton) and MNA score, the systolic OH₁, OH₁ and systolic OH₅ were statistically significant between severe sarcopenia and the robust group (OR 3.26, confidence interval [CI] 0.98–10.84; $P = 0.05$ for systolic OH₁; OR 4.31, CI 1.31–14.15; $P = 0.016$ for OH₁; OR 4.09, CI 1.01–16.55; $P = 0.048$ for systolic OH₅). Only systolic OH₁ was statistically different between sarcopenia and severe sarcopenia groups (OR 2.64, CI 1.87–8.73; $P = 0.012$). OH₁ and systolic OH₅ and OH₅ were statistically significantly different between severe sarcopenia and probable sarcopenia groups (OR 3.50, CI 1.34–9.11; $P = 0.010$ for OH₁; OR 3.24, CI 1.13–9.23; $P = 0.009$ for systolic OH₅; OR 3.50, CI 1.31–9.35; $P = 0.004$ for OH₅) (Table 2). All aforementioned ORs showed a positive correlation. There was no relationship between robust and probable sarcopenia after logistic regression analysis or between the other binary groups without logistic regression analysis ($P > 0.05$).

Table 1. Comparisons of patient characteristics, and OH₁, OH₃ and OH₅ according to sarcopenia stages

	Robust (n = 207)	Probable sarcopenia (n = 216)	Sarcopenia (n = 31)	Severe sarcopenia (n = 57)	P- value
Demographic features					
Age (mean ± SD)	72.57 ± 6.90	76.31 ± 6.75	77.10 ± 7.67	81.30 ± 6.33	<0.001**
Gender (female %)	66.2	63.0	96.8	94.7	<0.001**
Education year (median [IQR])	5 [0]	5 [1]	5 [3]	5 [0]	<0.001**
BMI (kg/m ²) (mean ± SD)	28.82 ± 5.00	29.51 ± 4.92	25.25 ± 3.38	24.93 ± 4.51	<0.001**
Comorbidities (%)					
Falls	33.3	43.5	38.7	68.4	0.003**
Dementia	11.7	27.6	33.3	33.9	<0.001**
Cerebrovascular disease	8.2	5.1	9.7	8.8	0.434
Peripheral vascular disease	7.2	8.8	9.7	5.3	0.400
Depression	44.4	45.8	51.6	57.9	0.450
Hypertension	59.9	70.4	58.1	73.7	0.092
Diabetes mellitus	30.4	30.1	29.0	24.6	0.845
Hyperlipidaemia	18.4	18.1	19.4	22.8	0.647
Coronary artery disease	19.3	19.0	25.8	21.1	0.859
Congestive heart failure	5.8	6.9	3.2	10.5	0.701
COPD	11.6	13.4	6.5	7.0	0.167
Hypothyroidism	18.8	23.6	25.8	21.1	0.705
Number of drugs (median [IQR])	4 [4]	5 [4]	5 [5]	7 [4]	0.016**
Class of drugs (%)					
ARBs	35.5	34.2	22.6	32.7	0.782
ACEIs	11.8	14.9	9.7	12.7	0.370
Beta-blockers	28.1	33.7	29.0	36.4	0.224
Calcium channel blockers	20.7	29.7	25.8	34.5	0.085
Diuretics	34.0	38.1	25.8	41.8	0.387
Alpha-blockers	10.8	8.4	3.2	7.3	0.409
Insulin	3.9	7.9	9.7	9.1	0.090
Antidepressants	34.5	41.1	22.6	49.1	0.170
Antipsychotics	3.9	6.9	0	14.5	0.184
Anti-Parkinson drugs	6.4	9.4	3.2	7.3	0.263
Laboratory findings					
Haemoglobin (g/dl) (mean ± SD)	13.02 ± 1.32	12.44 ± 1.38	12.33 ± 1.70	12.35 ± 1.18	0.001**
Glucose (mg/dl) (median [IQR])	98.50 [37]	99.50 [43]	98 [46]	103 [48]	0.725
Albumin (g/l) (mean ± SD)	4.08 ± 0.32	3.96 ± 0.35	3.99 ± 0.41	3.86 ± 0.36	0.001**
TSH (mg/dL) (median [IQR])	1.24 [1.15]	1.34 [1.16]	1.51 [1.29]	1.24 [1.15]	0.261
Vitamin B12(pg/ml) (median [IQR])	289 [209]	330 [259]	278 [310]	322 [547]	0.208
25(OH)D (ng/ml) (mean ± SD)	24.26 ± 12.29	22.85 ± 10.00	26.96 ± 17.79	29.99 ± 20.94	0.039**
Comprehensive geriatric assessment (median [IQR])					
MMSE	27 [5]	23 [6]	23 [7]	18 [6]	<0.001**
YGDS	2 [5]	3 [5]	3 [5]	4 [9]	0.017**
Basic ADLs	95 [10]	90 [15]	95 [25]	75 [30]	<0.001**
Instrumental ADLs	21 [6]	17 [5]	21 [7]	10 [13]	<0.001**
Tinetti-gait	12 [1]	11 [3]	12 [2]	9 [4]	<0.001**
Tinetti-balance	16 [2]	14 [4]	15 [2]	10 [6]	<0.001**
Tinetti-total	27 [3]	25 [7]	26 [4]	19 [8]	<0.001**
Up&Go Test	11 [4]	14 [8]	12 [6]	24 [15]	<0.001**
MNA	13 [2]	13 [2]	13 [5]	11 [2]	<0.001**
Orthostatic hypotension (OH) (%)					
Systolic OH ₁	14.0	19.9	9.7	31.6	0.037**
Diastolic OH ₁	6.8	9.7	12.9	14.0	0.303
OH ₁	15.9	25.0	22.6	38.6	0.011**
Systolic OH ₃	15.5	17.6	16.1	25.0	0.297
Diastolic OH ₃	7.7	7.9	12.9	12.5	0.433
OH ₃	19.8	20.4	29.0	28.6	0.199
Systolic OH ₅	14.5	17.6	19.4	32.1	0.071
Diastolic OH ₅	8.2	12.0	12.9	12.5	0.662
OH ₅	18.8	21.8	25.8	41.1	0.011**

25(OH)D, 25-hydroxyvitamin D; ACEIs, angiotensin-converting enzyme inhibitors; ADLs, activities of daily living; ARBs, angiotensin receptor blockers; BMI, body mass index; COPD, chronic obstructive pulmonary disease; IQR, interquartile range; MMSE, Mini-Mental State Examination; MNA, Mini-Nutritional Assessment; OH, orthostatic hypotension; OH₁, OH at the first minute; OH₃, OH at the third minute; OH₅, OH at the fifth minute; TSH, thyroid-stimulating hormone; YGDS, Yesavage Geriatric Depression Scale. *All odds ratios showed a positive correlation. **Bold values were considered statistically significant.

Table 2. The relationship between OH and different stages of sarcopenia by binary logistic regression analysis

	Exp (B)	95% CI	P-value
		Between the robust and probable sarcopenia group*	
OH ₁	1.34	0.78–2.31	0.278
		Between the severe sarcopenia and robust group*	
sysOH ₁	3.26	0.98–10.84	0.05
OH ₁	4.31	1.31–14.15	0.016
sysOH ₅	4.09	1.01–16.55	0.048
OH ₅	2.18	0.65–7.24	0.203
		Between the sarcopenia and severe sarcopenia group*	
sysOH ₁	2.64	1.87–8.73	0.012
		Between the severe sarcopenia and probable sarcopenia group*	
OH ₁	3.50	1.34–9.11	0.010
sysOH ₅	3.24	1.13–9.23	0.009
OH ₅	3.50	1.31–9.35	0.004

Exp (B), expected B; CI, confidence interval; OH, orthostatic hypotension; OH₁, OH at the first minute; OH₃, OH at the third minute; OH₅, OH at the fifth minute; sys, systolic *Independently age, body mass index, gender, education year, falls history, presence of dementia, Mini Mental State Examination, Yesavage Geriatric Depression Scale, Tinetti-gait and balance test, Tinetti total score, Up and go test score, Activities of Daily Living (Barthel and Lawton) and Mini Nutritional Assessment score. **Bold values were considered statistically significant.

Table 3. The relationship between muscle power, muscle mass, walking speed and OH

	Low muscle mass (%)	P-value	Low muscle pPower (%)	P-value	Low walking speed (%)	P-value
Sys OH ₁ (+/–)	30.1/22.2	0.107	68.8/57.4	0.546*	55.9/35.4	0.005*
Dia OH ₁ (+/–)	34.0/22.6	0.079	70.2/58.4	0.116	51.1/37.9	0.079
OH ₁ (+/–)	31.9/21.3	0.253*	71.6/55.9	0.168*	53.4/34.9	0.026*
SysOH ₃ (+/–)	36.0/20.9	0.053*	64.0/58.4	0.327	53.9/35.9	0.049*
Dia OH ₃ (+/–)	34.1/22.5	0.084	63.6/59.0	0.551	45.5/38.4	0.360
OH ₃ (+/–)	35.5/20.3	0.030*	62.7/58.5	0.424	50.0/36.0	0.060
SysOH ₅ (+/–)	37.0/20.6	0.024*	67.4/57.7	0.085	52.2/36.1	0.065*
Dia OH ₅ (+/–)	31.5/22.6	0.145	68.5/58.3	0.150	57.4/36.8	0.015*
OH ₅ (+/–)	37.6/19.3	0.019*	66.7/57.3	0.069	54.7/34.4	0.012*

Dia, diastolic; Exp (B), expected B; CI, confidence interval; OH, orthostatic hypotension; OH₁, OH at the first minute; OH₃, OH at the third minute; OH₅, OH at the fifth minute; Sys, systolic *P-value, adjusted for age, presence of dementia, fall history, Timed up and go score and Activities of Daily Living (Barthel and Lawton). **Bold values were considered statistically significant.

When the relationships between OH, and low muscle power, low walking speed and low muscle mass were assessed (adjusted for age, presence of dementia, fall history, Timed up and go score and ADLs scores), OH₃, systolic OH₅ and OH₅ were found to be associated with lower muscle mass ($P < 0.05$); and systolic OH₁, OH₁, systolic OH₃, diastolic OH₅ and OH₅ with lower walking speed ($P < 0.05$); however, OH was not associated with lower muscle power ($P > 0.05$, Table 3).

Discussion

Findings from the present study suggest that there is a close relationship between sarcopenia and OH, especially in the first and fifth minutes, which increases with the severity of sarcopenia. Moreover, decreased muscle mass and lower walking speed were found to be closely related to OH rather than to a decrease in muscle strength.

Sarcopenia, one of the geriatric syndromes, has attracted attention for the last three decades [17]. Although revised criteria for sarcopenia were published in 2019, the number

of studies using these criteria is small. In the present study, in which EWGSOP2 criteria were applied, 40% of the patients had no sarcopenia, whereas 11% had severe sarcopenia. Similar results were obtained in a study using EWGSOP1 criteria and including geriatric outpatients [20]. The high prevalence of sarcopenia is remarkable in older adults because sarcopenia is not only known as a simple muscle disease but is also known to affect many organ systems, particularly the respiratory and cardiovascular system.

Not surprisingly, this study found that the severity of sarcopenia/sarcopenia was associated with the development of OH, which can be explained by several mechanisms. First, sarcopenia can lead to a reduction in effective venous return, since venous pumps in the leg muscles pump blood from the lower extremity to the heart, which is important in maintaining cardiac filling pressure [21]. To date there is no other study investigating the relationship between sarcopenia and OH. However, some studies indirectly support the present findings. For example, Suzuki *et al.*, using a sample of young adults, reported that there was a decrease in venous return and cardiac output following 20 days bed rest, due to the decrease in muscle mass and muscle strength, and that

patients had a deterioration in orthostatic tolerance capacity [22]. In another study, it was found that calf circumference, which has a negative close correlation with appendicular skeletal muscle mass, could be used for OH screening in older adults [22]. Moreover, isometric contraction of the muscles under the waist, which play a role in maintaining blood pressure when one stands up by increasing venous return, is recommended for the prevention of OH [23,24]. This manoeuvre, which is well known for its effect on OH management, may actually indicate the importance of muscle mass and sarcopenia in the development of OH. In addition, in a study including 37 patients, sarcopenia was diagnosed by skeletal muscle index (SMI); lower systolic blood pressure was more common in those with, rather than those without, sarcopenia. However, there was no correlation with OH, which may be due to the small sample size of the study [25]. The results of these studies are consistent with the present findings, which clearly reveal the relationship between sarcopenia and OH, suggesting that it is a consequence of a reduction of skeletal muscle mass rather than muscle strength. According to findings from this study, skeletal muscle pump activity that provides venous return is an important factor in maintaining systolic blood pressure regulation, and the main factor determining this is muscle mass. Thus, sarcopenia seems to affect not only the muscular system but also cardiovascular functions. As a result, sarcopenia may impair the compensatory response to orthostatic changes in older adults, leading to early (1 min) and delayed (5 min) OH development after standing up [26]. Although the cause is not known exactly, OH in the first minute is shown to be more important for geriatric practice in older adults [25]. For example, in a study using HUT similar to that in this study, a relationship was found between frailty and OH only in the first minute, but sarcopenia was not evaluated in that study [14]. In another study, Juraschek *et al.* suggested that OH should be assessed within 1 min of standing, as it was found that OH₁ was most strongly related to dizziness and individual adverse outcomes including fall, fracture and syncope [27]. Therefore, sarcopenia may also have contributed to the development of OH and the outcomes in the first minute in those studies. According to the present findings, decreased muscle mass associated with OH in the fifth minute rather than in the first and the third minute is also noteworthy. It is possibly an indication that venous blood return cannot be sustained by muscle mass while standing. Moreover, in older adults, OH may play a role in the reduction of muscle mass and sarcopenia by causing falls, balance and gait problems, and possibly making patients less mobile [3,5]. Indeed, in this study, falls history, a decrease in gait and balance functions evaluated by Tinetti test, and a decrease in functionality evaluated by BADL and IADL were frequent, specifically in the severe sarcopenia group. However, further longitudinal studies are needed to determine the relationships between sarcopenia and OH.

The present study has several strengths. For the diagnosis of OH, the gold standard method, HUT test, and for the diagnosis of sarcopenia, EWGSOP2 criteria, were used.

Other strengths include the provision of adequate sample size and the evaluation of other risk factors, such as comorbid diseases and medication. However, findings should be interpreted in light of the study limitations. First, this study is cross-sectional in nature. Therefore, it is not known whether OH leads to sarcopenia or whether sarcopenia leads to OH. It is possible that the relationship is bidirectional. Thus, further longitudinal studies are now required for the determination of causality. Secondly, because there were no data on inflammation and oxidative stress makers, heart rate variability or brachial–ankle pulse wave velocity, the mechanism underlying the association between sarcopenia and OH cannot be clarified. The blood pressure data beyond 5 min of HUT were not collected and it is therefore unclear how the results would be affected by prolonged standing. In addition, the exclusion of older patients due to the contraindications for HUT is a limitation. Lastly, 30 min may not be an adequate length of time to avoid the effects of smoking, consuming caffeine and exercising prior to HUT.

OH and sarcopenia are two major geriatric syndromes that are closely related. Moreover, the greater the severity of sarcopenia, the stronger the relationship is to OH. Therefore, when evaluating older adults with sarcopenia in geriatric practice, OH should also be evaluated, and vice versa. Thus, more effective management of the two will be possible and common complications due to both may be reduced.

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